Response

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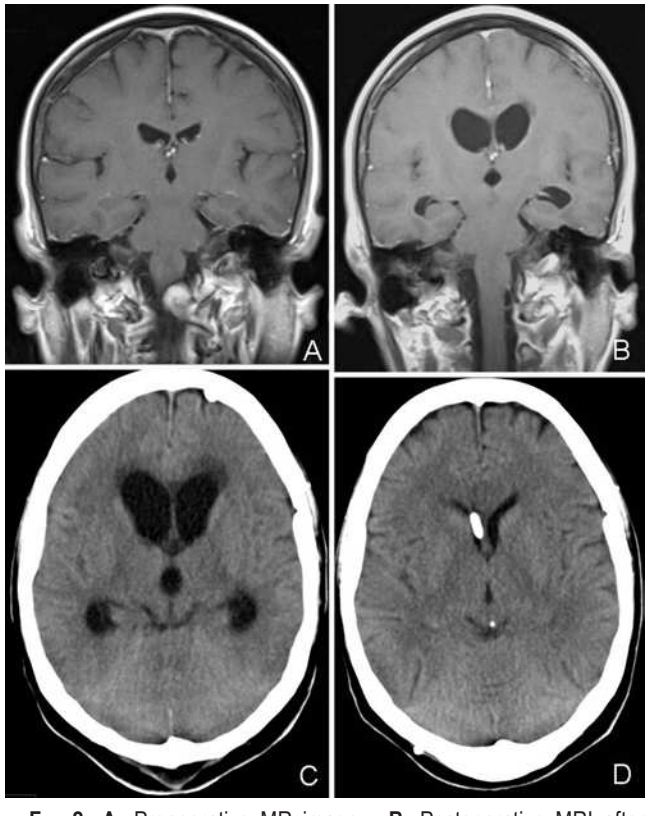

Fig. 2. A: Preoperative MR image. **B:** Postoperative MRI after meningioma removal. **C:** Axial CT scan demonstrating hydrocephalus 2 months after meningioma surgery. **D:** Axial CT scan after shunt insertion.

So, in this case, cell aggregations could represent a cofactor in hydrocephalus development. Surely a large study is necessary to highlight the prevalence of this finding in patients' CSF and to correlate cellular aggregate with hydrocephalus.

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Disclosure

The authors report no conflict of interest.

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Response: We thank Dr. Rossetto and colleagues for their interest in and comments on our recently published article on the occurrence of hydrocephalus after skull base meningioma surgery. The authors additionally report an unexpected CSF finding in a patient after resection of a foramen magnum meningioma with postoperative hydrocephalus. Aggregates of tumoral meningioma cells were found in the CSF after lumbar puncture 2 months postoperatively, and Rossetto et al. suggest the latter as an etiology for the occurrence of hydrocephalus. However, ventricular CSF analysis during ventriculoperitoneal shunting did not reveal any meningioma cells.

We agree that meningioma cell spread as etiology for hydrocephalus after meningioma surgery is an intriguing hypothesis, but it requires further investigation in a larger patient cohort. Spinal meningioma cell spread or spinal drop metastasis is not a new finding and is a well-known complication of intracranial meningiomas, especially when located in the posterior fossa, with a transitional histological characteristic in benign meningiomas or in high-grade meningiomas.[2] Similar to other CNS tumors with CSF spreading or intracerebral bleeding such as SAH, cells or blood products may follow the CSF path in a sedimentation manner. Although rare, data on benign meningiomas (WHO Grade I) showed intracerebral spreading besides a spinal or extraneural spreading.[1] In this study, none of these patients were symptomatic with hydrocephalus. Therefore, detection of meningioma cells in CSF may not necessarily be a reason for impairment of CSF circulation.

Nevertheless, Rossetto et al. present an interesting case, and further studies should address if meningioma cell spreading and aggregation sufficiently explain the etiology of hydrocephalus in meningioma patients.

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